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Conference Paper · September 2013

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A cellular automata to model epidemics.

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Abstract:

Compartmental models are very popular in epidemiology (8), they provide excellent results when the populations satisfy certain hypotheses as large population size or population homogeneity (11), the complexity of this models is low making their analysis intuitive. In the other hand, they ignore important factors inherent to the problem, such as the nature of contacts between individuals and population heterogeneity (4 5 18). Cellular automata models are adequate to describe natural systems consisting of a massive collection of simple objects. They represent the global system behavior as a colection of simpler objects or cells. In this paper we propouse a cellular automata model to study the time evolution of a heterogeneous population through the various stages of disease resulting from the individuals interactions (epidemic). We validate the model with real data of flu that hit Geneva (Switzerland) in 1918 and then we will test the model under different assumptions discussing the result that each has on the disease dynamics.

key words:

cellular automata, modell, epidemics, heterogeneity, individual based model, public health.

Introduction:

Actually many of existing epidemic models employ differential equations (8) and do not take into account of spatial factors such as variable population density and population dynamics (4, 5, 9, 19). This kind of models also incorporate the homogeneous mixing assumption, whereby the rate of increase in epidemic incidence is proportional to the product of the number of infectious and susceptible individuals. Some authors have been relaxed this assumptions , but not eliminated of their models (10, 13, 15, 18). The homogeneous mixing assumption is robust in the sense that it is consistent with several scenarios for the individual-to-individual transmission of disease. In particular, it is equivalent to a model in which all individuals in a population make contact at an identical rate and have identical probabilities of disease transmission to those contacts per unit of time. Although this assumption is unrealistic, it facilitates mathematical analysis and, in some cases, offers a reasonable approximation.

In the real world, populations are heterogeneous in some aspects such as: susceptibility, infectiousness, contact rates or number of partners, and simple homogeneous mixing models do not allow for extreme variation in host parameters. Heterogeneity in susceptibility and infectivity is an important feature of many infectious diseases and has been considered to improve the accuracy of epidemiological models. The focus has been placed on the impact of heterogeneity in the final size of epidemics as well as on its consequences to disease control, and data interpretation (4, 5, 21). It has been shown that the final size of the epidemic is reduced when the risk of infection is heterogeneously distributed, both for the deterministic and the stochastic formulations. Later, results were extended to the investigation of epidemic spread in a random network.

Actually there are models that capture some, but not all, of these features (7, 12, 20). An epidemic model should incorporate aspects like: individuals had contact with only a finite number of other individuals in the population at any time, and contacts that can result in disease transmission are usually short and repeated events; the number and frequency of contacts between individuals is not homogeneous; the numbers and identities of an individual's contacts will change as time goes by and the individuals have potential to transmit the pathogen and susceptibility.

Cellular automata models (*CA*) can fill these aspects and have been used by several researches as an efficient alternative method to simulate epidemic spreading. A two-dimensional CA is formed by a two--dimensional array of identical objects called *cells*, which are endowed with a state that changes in discrete steps of time according to specific rules. An updated function determines how local interactions, can influence the global behavior of the system (1, 6, 14). It is of special interest the CA-epidemic proposals modeling the motion of individuals.

In the current literature there are many implementations of automata based models (14, 16, 17, 22). The ways of approaching the modeling are diverse and can be grouped into different categories according to model, continuous or discrete space, time or individuals.

Actually there are many works in which each cell is considered as a homogeneous distribution of individuals, represent areas of equal size containing a specific population (16, 17, 22). Different cells have different densities and possibly different mobility properties. Infection occurs through contact between individuals of the same cell or neighboring cells. Differential equations are explicitly included in cells, and the evolution of the epidemic in each temporal evolution follows the classical model, with the modifications that arise from the passage of individuals from a particular cell to another. However, taking all the automata as a whole, the temporal evolution of the epidemic is similar to the classical model. Many models use deterministic rules for the upgrade on of the cells. But in the case of epidemics, probabilistic rules seem to reflect more real behavior.

In order to address these issues, and develop and validate a model for an real epidemic situation (8), we introduced an *individual-based-model* built upon cellular automata that include all the features described before. This allows us to capture the individual heterogeneity as well as a realistic model of individual contacts, modeling individuals explicitly exposed, on the other hand, relaxing the assumptions of classical models allows us to evaluate different scenarios and strategies for action. Each individual will be characterized by its own intrinsic infectivity, expressed as the expected reproductive number R_i, which will be achieved by varying the infectiousness (i.e., the rate of transmission, given an unlimited supply of susceptible) between individuals. Different assumptions with which we construct the classical model are applied directly to each individual, and the differential equations of the classical model are incorporated implicitly through rules.

In this paper we first describe how we implement a individual based model build on a cellular automata approach, later, with the validated model, we implement different strategies and analyze some possible epidemic scenarios.

Work elements and methodology:

Our model is based on cellular automata and build on the information collected about pandemic flu defect to Swiss canton of Geneva in the early twentieth century and modeled before by other authors (2, 3, 8).

A first definition of the model is:

- Each cell represents an individual in one of the possible states, or the state of empty cell. No distinction is made between the state of the deceased and the empty cell. Births involve passing empty cell to a susceptible cell.
- The transition between states is probabilistic.The initial spacial distribution is random, provided the assumption of homogeneous distribution for large population sizes and thus validate the classic approach.Other spatial distributions can be used as seen below.

- At first it simulates a random motion of the automaton through a reciprocal change in state neighboring cells, ie, a cell goes from X_i to X_j status as a neighbor state changes from E_j to E_i. This movement seems to emulate the approximate movement of real people (who really do not follow random movements), which contributes to the homogeneous distribution and contacts between infectious and susceptible. Other types of movements can be simulated to model different situations as we see later.
- Potentially infectious contact is made between infectious individuals and susceptible within the neighborhood defined as a zone of influence.
- For simplicity, the grid type used is rectangular, with Moore neighborhood, with variable size.
- The boundary condition is fixed, with a contour consisting of non interacting empty cells, compatible with the situation in a city, an area of high population density surrounded by a much lower density area.
- The simulation progresses in discrete time *t* given by $t = 1/dt$, with $dt=1,2,...,n$.

Each cell is then defined as a stochastic Moore machine (2 4) by A = (E, X, Y, δ), where:

- E, the set of possible states comprises *6* states: *S* (susceptible), *E* (exposed), *I* (infectious), *A* (asymptomatic), *R* (recovered) and *D* (Removed or empty).
- X is the set of input (real numbers). An automaton receives input only when *E=S*, issued by another with *E=I* or *E=A*, when the automaton is in the vicinity of the issuer. Transitions that do not involve contact with infectious individuals are made in probabilistic form independently of a possible entry (transitions on empty entry ε).
- Y is the output set of X issued in state *I* or *A*, corresponding to the input received in state *S*. The output corresponds to the infection probability from contact that has the automata in stage *I* or *A*, obtained from distributing the β value for that automaton in the neighborhood under consideration.
- δ is the state transition function, which applied to the active state at iteration *k*, the state decides probabilistically active at iteration *k +1*. The function is applied in two steps, one for the state change and recovery from infection and the other corresponding to the movement. To decide the status changes define two probability matrices: one for empty transition ε and a empty entry for the transition from contact with infectious.
- For each element of the matrix p_{ij} the probability of moving from state *i* to *j* in each time step, and placing the states *S, E, I, A, R* and *D* in increasing order from row or column 1 to 6, is defined the transition matrix for empty entry(see Table1)

	S	E	I	A	R	D
S	1−μ	β	0	0	0	μ
E	0	1−(ε+μ)	ερ	ε(1−ρ)	0	μ
I	0	0		0	γ1	μ
A	0	0	0		γ1	μ
R	0	0	0	0	1−μ	μ
D	μ	0	0	0	0	1−μ

Table 1: Transition matrix for empty entry

Defining the size of the neighborhood as ν and the input value as λ, the contact transition matrix is defined in table 2

	S	E
S	$1-(\lambda/\nu)$	λ/ν
E	0	1

Table 2: contact transition matrix

The individuals movement is equally likely from a cell centered in an area of predefined size to any other within in the area. The cells swapped positions, which can be interpreted as changes of state. The output function gives the value of infection rate if the automaton is in state I or *A*. The initial state vector $(P(o))$ is composed by the probabilities for each initial state given for the automaton. defined as the total number of cells in the grid and as $P(o)=[S_i/G, E_i/G, I_i/G, A_i/G, R_i/G, D_i/G]$. An interesting aspect of the model is that it allows two different ways to incorporate an extra state. Transfer to Reported can be done in a probabilistic sense or can be deterministically as in classical models

Now, the *CA* is defined by *R*=*G*(*T*,*C*) where:

- The topology *T* is square. The neighborhood is kind Moore, and is only seen for cells in stage *I* or *A*. The boundary conditions are fixed, with a outline consisting of empty cells not interacting.
- The connection *C* is unidirectional from cells in state *I* or *A* to the cells in a state *S* that are in the neighborhood. The connection is isotropic and uniform anywhere in the neighborhood, and provides an entry for each cell in *S* consisting of the value of □ that has the cell *I* or *A*, used to make the transition to the probabilistic state *E*. The cells in a state *S* which are included in several neighborhoods in a given time step will have many opportunities to change state as the number of neighborhoods in which they are included.

We can see all the illness process; first, during infection, if the individual is in infectious state *I* checks the availability of susceptible neighbors with whom to contact if there is indeed likely in the neighborhood and the likelihood of the event of infection occurs is fulfilled then the neighbor state changes to stationary or latency *E*. For individuals in asymptomatic state the behavior is exactly the same changing the infection probability (algorithm 2).

In the transition from exposed to infectious behavior is summarized check two different probabilities, if the transition probability satisfies the infectious state, the individual become infectious (*Status* = *I*), on the other hand, if the probability is met transition to asymptomatic, then the individual becomes asymptomatic (*Status* = *A*). This is true provided that the individual current is in steady state (*state* = *E*) (Algorithm 3).

During the recovery phase if the individual is in infectious state (*Status* = *I*) or asymptomatic state (*Status* = *A*) and satisfies the likelihood of recovery, then it passes to recovered state (*Status* = *R*).
In death by illness, for infectious individuals that satisfies the probability of death, then goes to dead state (*state* = *D*) or empty (Algorithm 4).

Death from disease is probabilistic, this probability was adjusted according to the actual data, if an individual meets the probability of death by disease is removed, leaving an empty cell in the grid (Algorithm 5).

In the case of natural deaths, if the current cell is not empty and satisfies the probability of natural death, then goes to dead or empty state (*State = D*), if the cell is in empty, and holds the probability of birth, then it switches to susceptible state (*Status = S*). The odds of births and natural deaths are equal, so this process does not affect in the first instance the size of the population (Algorithm 6).

Finally, in the movement phase, two cells exchange their values, first should generate a random perturbation to determine the direction of motion of the individual (when using random motion), then the current state of the individual is stored in an auxiliary variable, the current status of the individual becomes the state of the cell to the moving and the state of the adjacent cell becomes stored in the auxiliary variable, this process is repeated at each position in the grid and an individual can move more than once in the same cycle (Algorithm 7).

The algorithm explaining the dynamics of the disease can be seen below, may be seen in the order of the steps previously described (Algorithm 1) and each step is described in Appendix A.

Some of the advantages of this model are:

- Allows a greater degree of heterogeneity in modeling each individual as a cell within the grid.
- Allows modeling different spatial distributions, which can result areas most densely populated and less dense areas, resembling the grid topology to that of a real city.
- Directed movement can be tested to generate different topologies grid, although is not very realistic, this allows to evaluate how the spatial distribution affects the temporal dynamic.
- Situations as quarantine or other preventive measures can be modeled simply to assess the effects on the evolution of the disease.
- The relaxation of the assumptions of the classical model allows us to evaluate different control strategies and then assess their impact on the dynamics.

For adjustment we must consider several points that arise due to the approach. First, there is the problem of scale to be used, the larger the grid and employed population, the closer we get to the large population assumption of the classical model and the computational cost increases, is non desirable to use grid sizes too big for temporal behavior analysis of the epidemic, but it is for the purpose of validating the model with parameters of the classical model.

The size of the neighborhood, determines the degree of global influence. The larger the neighborhood size used, the closer the results to the assumption of spatial homogeneity of classical model. This is because a very large neighborhood can influence the infectious even in low density areas of infectious ,"*softening*" the temporal dynamics.

We modeled the infectiveness of each individual using a bimodal probability distribution with two modes around which are distributed two populations of infective individuals, one being more active than the others. In figure 1 we show the resulting adjusted model comparing it with clasical model, this shows that the model can explain the real dynamics of a real epidemic, the adjusted model is then used to evaluate different hypotheses.

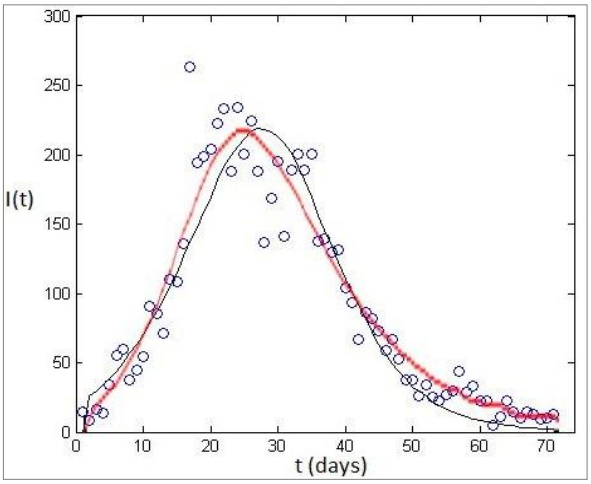


Figure 1: Dynamics of a real epidemic, the data is from 1918 Spanish Flu.

° Real Data
- Clasic mode adjustment, Error aproximation ~ 8%
- Cellular Automata adjustment, Error aproximation ~ 8%

Algorithm 1: Cellular automata dinamics

```
Grid{NxN square grid where the automata evolve}
Nt = T otalIndividuals
N = T otalDays
for Day = 1 → N do
        for i = 1 → Nt do
                {Infection, Algorithm 2}
                {Exposed to Infectious, Algorithm 3}
                {Reported, Algorithm 4}
                {Recovery, Algorithm 5}
                {Dead by illness, Algorithm 6}
                {Births and natural deaths, Algorithm 7}
                {Individuals movement, Algorithm 8}
        end for
end for
```

Results and discusion:

With all this considerations, our model allows us to propose different scenarios to assess the dynamics of a disease process and thus determine how each factor influences the dynamics.

To approach to realistic conditions, simulations were performed with real population size (*N=75.000*), and a neighborhood around the grid size ($r = 3$) using adjusted parameters from the classical model. The initial distribution of individuals is kept uniform. As we indicate before, the infection rate, to take into account the role of individual variation in the infection process. is can be described using a probability density function, for the case a bimodal distribution with two modes: β_i and β_s for the probability of infection through contact with diferent kind of infected individuals was used; also present asymptomatic individuals, who have a very low infectivity but influence the dynamics of the epidemic as they represent a significant percentage of the population ().

Another assumption made in population models is that of spatial homogeneity. But if the epidemic began in one or a few bounded regions, the homogeneity could not be achieved instantly if we assume random movement of individuals. Varying the initial spatial distribution of infectious individuals shows how the behavior of the epidemic is distancing itself more than expected by . In the first case, initially infectious individuals occupy one half of the grid. The evolution reaches lower values of infectious because, being more confined to an area of the grid, infectious encounters with others are more infectious than expected in homogeneous conditions. In the second quarter of the grid and in ther third case eigth of the gride size is fill with infetious individuals. In these cases, as we show in figure 2 the epidemic remains at a low but persistent, to not having enough susceptible individuals in the neighborhood at first, but finding new susceptible as the movement is made at random.

Making a new test for the third case but directed movement (from one corner to the rest of the grid) show a partial recovery of the expected behavior but delayed several days. This is expected to recover homogeneous state more quickly thanks to the movement is not random.

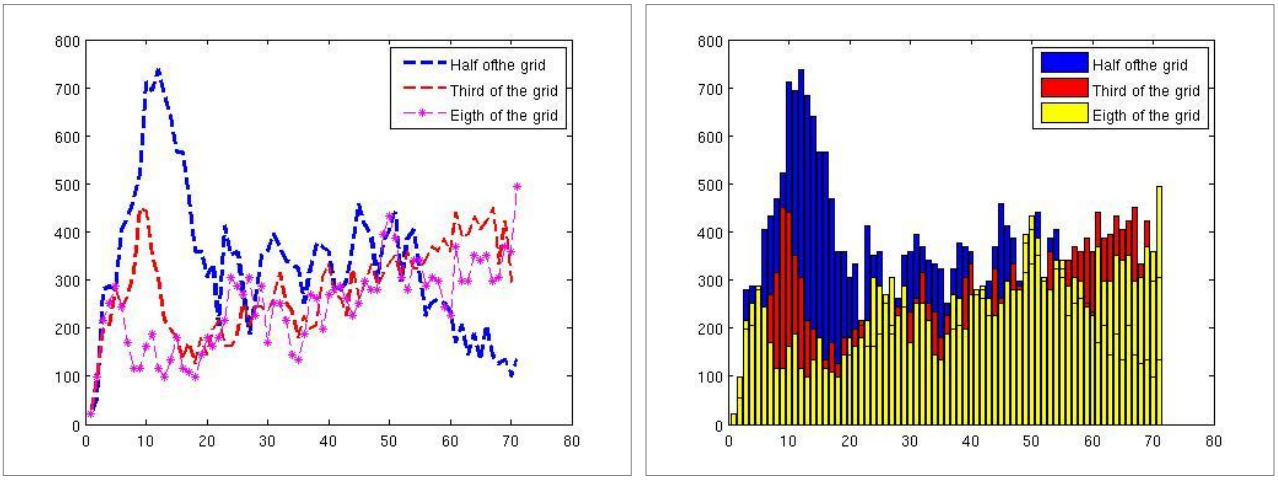


Figure 2: initial spatial distributions for infected individuals.

A mixed strategy, for example if we take into account a medium density population and that a percentage of it is moving an urban center for a certain period of time, while the rest of the population remains in peripheral areas; this scenario tries to emulate the situation of a real city in which we can find a densely populated urban center surrounded by a peripheral area of lower concentration of population; the dynamics in this case are show in figure 3-a the number infected is triggered at the beginning of the epidemic, this is because a large number of individuals are infected due to high conentración in the center making more easyt the transmission, in figure 3-b can be observed behavior for the case that the distribution was uniform individuals, in this case the dynamics uniform in time, infected population do not change drasticaly at time *t*.

Another strategy that is typically used to control an epidemic is vaccination, to assess their effect on the temporal dynamics different populations proposed initial immune individuals, as shown in figure 4 the effect of vaccination plan for an urban population with high population density is a drastic decrease in the number of new cases this is because the initial susceptible population level is considerably lower, in this way, the infected individuals on average have less contact with susceptible, reducing the reproductive number for each generation.

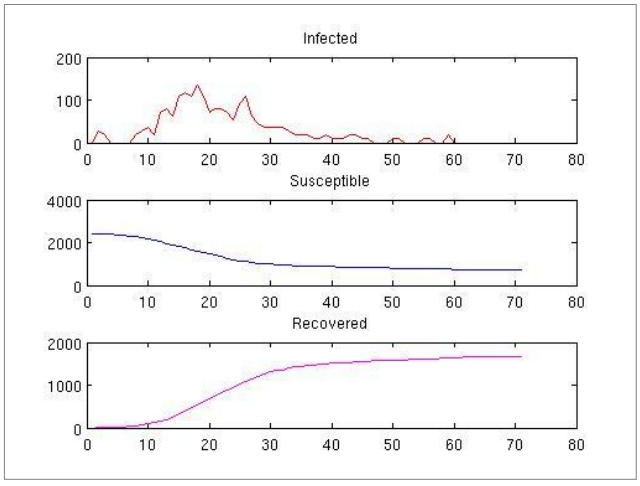


Figure 3-a: Dynamics for high concentration spatial distribution.

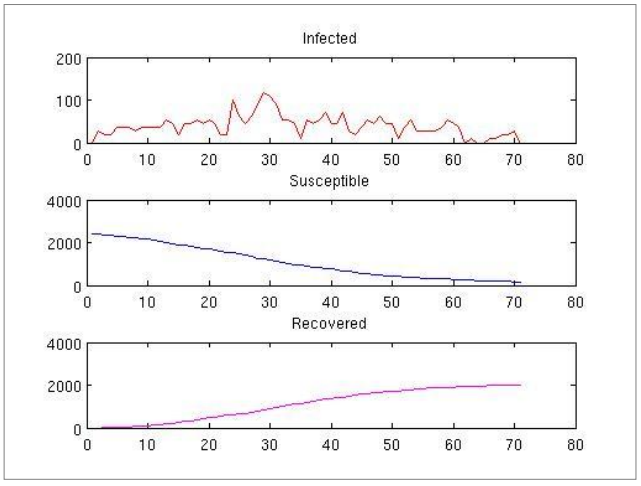
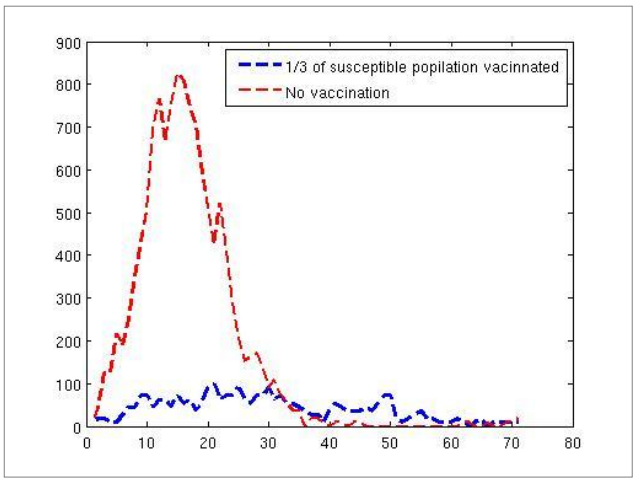


Figure 3-b: Dynamics for uniform concentration spatial distribution and low density.



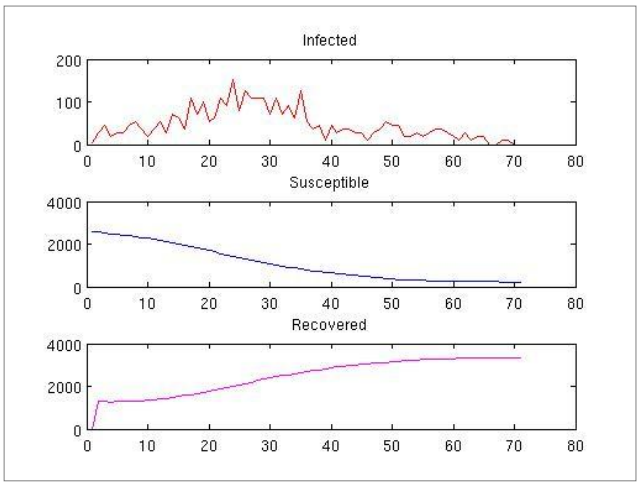


Figure 4: vaccination effect in disease dynamics; left: – no vaccination, – 1/3 of susceptible population vacinnated; rigth: dynamics with vaccinated population for infected, susceptible and recovered indiviauduals

Finally the last scenario that is a quarantine, this strategy is usually analyzed in other works, is expected to limit the movement of people after a certain period affect disease dynamics in our model population stops moving after the number of infected individuals reaches or exceeds a certain threshold (1%, 3%, 5% of population infected), the results of this can be seen in figure 6 Reduce the mobility of individuals seems to work much better when you have high dense populations, such populated urban centers, this can be because in areas of lower population density the quarantine is located in some way implicit and contact between individuals is less frequent.

The implementation of different probability distributions for the population of infectious individuals results in greater heterogeneity and a dynamic that is more like the real one. The simulation of random motion through the reciprocal exchange of states between adjacent cells contributes the

homogeneous distribution of the population as is assumed in classic modeling, which increases the probability of contact between individuals susceptible and infectious and therefore the spread of the epidemic. In the other hand, different spatial distributions can show different temporal dynamics, evualuating how much the spatial dsitribution affects this aspect.

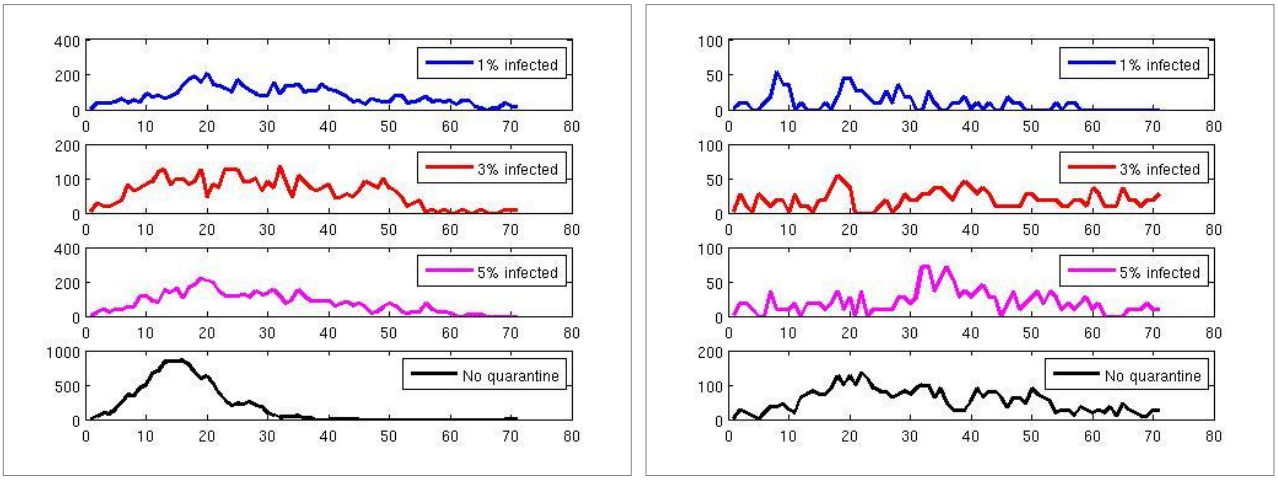


Figure 5: Effect of quarantine on the dynamics of the disease; left: quarantine at a high population density; right: quarantine at a low density population.

Finally it can be observed that implement control strategies such as vaccination of a proportion of the population or quarantine reduces the effect of the epidemic, in the second case the strategy works if it detects the danger in time, and the population density is very high , for the case of vaccination may observarese the reproductive number decreases considerably siginificativo vaccinating a number but not necessarily too high (about 30% of the susceptible population) individuals.

The model implemented in this work to evaluate different scenarios and control strategies by relaxing the assumptions of the classical models can deal with cases such as populations of small or medium size, on the other hand, the rules that define stochastic cellular automata to assess a large number of strategies related to the distribution and spatial movement of individuals and its effect on disease dynamics.

Acknowledgments
The Authors whishes to thanks: The Littoral National University (*UNL*) and the National Council of Scientific and Technological Research (*CONICET*) from Argentina for their support.

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Appendix A: Algorithms.

Algorithm 2: Infectious state

```
if State = I then
        if Neighbor = S then
                Z ~ U[0, 1]
                if Z < β/ν then
                        State = E
                end if
        end if
else
        if State = A then
                Z ~ U[0, 1]
                if Z < qβ/ν then
                        State = E
                end if
        end if
end if
```

Algorithm 3: Exposed state

```
if State = E then
        Z ~ U[0, 1]
        if Z < εr then
                State = I
                if Z < r then
                        State = A
                end if
        end if
end if
```

Algorithm 4: Recovery step

```
if State = I or State = A then
        Z ~ U[0, 1]
        if Z < γ1 then
                State = R
        end if
end if
if State = J then
        Z ~ U[0, 1]
        if Z < γ2 then
                State = R
        end if
end if
```

Algorithm 5: Dead by illness

```
if State = I or State = J then
        Z ~ U[0, 1]
        if Z < δ then
                State = D
        end if
end if
```

Algorithm 6: Births and natural deaths	**Algorithm 7:** Individuals movment

if State D **then**
Z1 ∼ U[0, 1]
Z2 ∼ U[0, 1]
if Z1 < μ **then**
State = D
end if
if Z2 < μ **then**
State = S
end if
end if
	Z1 , Z2 ∼ U[−r, r]
	Aux = State(i, j)
	S tate(i, j) = State(i + Z1 , j + Z2)
	S tate(i + Z1 , j + Z2) = Aux

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